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     (FILE 'HOME' ENTERED AT 08:57:11 ON 28 JUN 2004)

     FILE 'CAPLUS' ENTERED AT 08:57:21 ON 28 JUN 2004
                E AJA T/IN
L1            1 S E4
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                SET DETAIL OFF

     INDEX 'HCAPLUS, WPINDEX, INPADOC' ENTERED AT 08:59:35 ON 28 JUN 2004
                SEA WO 2002070544/PN,APPS
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               1    FILE HCAPLUS
               1    FILE WPINDEX
               1    FILE INPADOC
L2              QUE WO 2002070544/PN,APPS
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     FILE 'HCAPLUS' ENTERED AT 08:59:38 ON 28 JUN 2004
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L4          SEL L3 1- PN APPS :       5 TERMS
L5            1 FSO L3
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                SET HIGHLIGHTING DEF
                SET NOTICE LOGIN DISPLAY

     FILE 'MEDLINE' ENTERED AT 09:00:49 ON 28 JUN 2004
                E AJA T/AU
L6            2 S E3
L7            1 S E4
L8            2 S E5
                E CHING B W/AU
L9            3 S E6
L10           1 S PROTEASE INHIBTORS
L11       23574 S PROTEASE INHIBITORS
L12           0 S PRESERVING ANTIGNS
L13           0 S PRESERVING ANTIGENS
L14         210 S PRESERVING AND ANTIGENS
L15           0 S L11 AND L14
L16           1 S PRESERVING VIRUS
L17         168 S PRESERVING AND VIRUS
L18           2 S L11 AND L17
L19        9329 S ANTIGEN PRESENTATION
L20          72 S L11 AND L19
L21          12 S VIRUS AND L20
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d l1 all

L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:696003 CAPLUS
DN 137:215798
ED Entered STN: 13 Sep 2002
TI Anti-apoptotic agents or interleukin 1β converting enzyme (ICE/CED-3) inhibitors for preserving antigenicity of markers associated with diseases
IN **Aja, Teresa**; Ching, Brett W.; Gladstone, Patricia L.
PA Idun Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 148 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07K
CC 15-1 (Immunochemistry)
Section cross-reference(s): 1, 7
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002070544	A2	20020912	WO 2002-US7208	20020301
	WO 2002070544	A3	20030821		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	US 2003039661	A1	20030227	US 2002-87607	20020301
PRAI	US 2001-272750P	P	20010302		

OS MARPAT 137:215798
AB The present invention relates generally to programmed cell death and specifically to methods, compns., and kits for preserving or enhancing antigenicity of markers associated with disease by utilizing inhibitors of apoptosis including interleukin-1β-converting enzyme (ICE)/CED-3 family inhibitors.
ST apoptosis caspase inhibitor disease marker immunogen antigenicity preservation
IT Antigen presentation
Cytomegalovirus
Drug delivery systems
Hepatitis virus
Human herpesvirus
Human immunodeficiency virus
Infection
Leukocyte
Neutrophil
Polymorphonuclear leukocyte
(anti-apoptotic agents or interleukin 1β converting enzyme (ICE/CED-3) inhibitors for preserving antigenicity of markers associated with diseases)
IT Antisense oligonucleotides
Nucleic acids
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(anti-apoptotic agents or interleukin 1β converting enzyme (ICE/CED-3) inhibitors for preserving antigenicity of markers associated with diseases)

=> d l21 1-12 all

L21 ANSWER 1 OF 12 MEDLINE on STN
AN 2004034908 MEDLINE
DN PubMed ID: 14734740
TI Escherichia coli expressing recombinant antigen and listeriolysin O stimulate class I-restricted CD8+ T cells following uptake by human APC.
AU Hu Paul Q; Tuma-Warrino Renee J; Bryan Marianne A; Mitchell Kathleen G; Higgins Darren E; Watkins Simon C; Salter Russell D
CS Department of Immunology and Cell Biology, University of Pittsburgh School of Medicine, Pittsburgh, PA 15213, USA.
NC CA073743 (NCI)
T32 CA082084 (NCI)
SO Journal of immunology (Baltimore, Md. : 1950), (2004 Feb 1) 172 (3) 1595-601.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200405
ED Entered STN: 20040122
Last Updated on STN: 20040510
Entered Medline: 20040507
AB Vaccination against cancer or intracellular pathogens requires stimulation of class I-restricted CD8(+) T cells. It is therefore important to develop Ag delivery vectors that will promote cross-presentation by APCs and stimulate appropriate inflammatory responses. Toward this goal, we tested the potential of Escherichia coli as an Ag delivery vector in in vitro human culture. Bacteria expressing enhanced green fluorescent protein were internalized efficiently by dendritic cells, as shown by flow cytometry and fluorescence microscopy. Phenotypic changes in DC were observed, including up-regulation of costimulatory molecules and IL-12p40 production. We tested whether bacteria expressing recombinant Ags could stimulate human T cells using the influenza matrix protein as a model Ag. Specific responses against an immunodominant epitope were seen using IFN-gamma ELISPOT assays when the matrix protein was coexpressed with listeriolysin O, but not when expressed alone. THP-1 macrophages were also capable of stimulating T cells after uptake of bacteria, but showed slower kinetics and lower overall levels of T cell stimulation than dendritic cells. Increased phagocytosis of bacteria induced by differentiation of THP-1 increased their ability to stimulate T cells, as did opsonization. Presentation was blocked by proteasome inhibitors, but not by lysosomal **protease inhibitors** leupeptin and E64. These results demonstrate that recombinant E. coli can be engineered to direct Ags to the cytosol of human phagocytic APCs, and suggest possible vaccine strategies for generating CD8(+) T cell responses against pathogens or tumors.
CT Check Tags: Human; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Antigen Presentation: GE, genetics
Antigen Presentation: IM, immunology
Bacterial Toxins: BI, biosynthesis
Bacterial Toxins: GE, genetics
*Bacterial Toxins: IM, immunology
*CD8-Positive T-Lymphocytes: IM, immunology
CD8-Positive T-Lymphocytes: ME, metabolism
CD8-Positive T-Lymphocytes: MI, microbiology
Cell Line
Cells, Cultured
Cysteine Endopeptidases: PH, physiology

Dendritic Cells: EN, enzymology
*Dendritic Cells: IM, immunology
Dendritic Cells: ME, metabolism
Dendritic Cells: MI, microbiology
*Escherichia coli: GE, genetics
Escherichia coli: GD, growth & development
*Escherichia coli: IM, immunology
Gentamicins: PD, pharmacology
*HLA-A2 Antigen: IM, immunology
Heat-Shock Proteins: BI, biosynthesis
Heat-Shock Proteins: GE, genetics
*Heat-Shock Proteins: IM, immunology
Influenza A Virus, Human: GE, genetics
Influenza A Virus, Human: IM, immunology
Kanamycin: PD, pharmacology
Kinetics
Luminescent Proteins: GE, genetics
Luminescent Proteins: ME, metabolism
Lymphocyte Activation: GE, genetics
Multienzyme Complexes: PH, physiology
Phagocytosis: GE, genetics
*Phagocytosis: IM, immunology
Recombinant Proteins: BI, biosynthesis
Recombinant Proteins: IM, immunology
Viral Matrix Proteins: BI, biosynthesis
Viral Matrix Proteins: GE, genetics
*Viral Matrix Proteins: IM, immunology

RN 147336-22-9 (green fluorescent protein); 59-01-8 (Kanamycin); 72270-41-8 (hlyA protein, Listeria monocytogenes)

CN 0 (Bacterial Toxins); 0 (Gentamicins); 0 (HLA-A2 Antigen); 0 (Heat-Shock Proteins); 0 (Luminescent Proteins); 0 (Multienzyme Complexes); 0 (Recombinant Proteins); 0 (Viral Matrix Proteins); 0 (influenza **virus** membrane protein); EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.25.1 (proteasome endopeptidase complex)

L21 ANSWER 2 OF 12 MEDLINE on STN

AN 2002475343 MEDLINE

DN PubMed ID: 12237893

TI Proteasome inhibitors reconstitute the presentation of cytotoxic T-cell epitopes in Epstein-Barr **virus**-associated tumors.

AU Gavioli Riccardo; Vertuani Simona; Masucci Maria G

CS Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, Sweden.

SO International journal of cancer. Journal international du cancer, (2002 Oct 20) 101 (6) 532-8.
Journal code: 0042124. ISSN: 0020-7136.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200211

ED Entered STN: 20020919
Last Updated on STN: 20021213
Entered Medline: 20021104

AB EBV-infected cells and EBV-associated tumors may evade CTL recognition by defective antigen processing, resulting in poor presentation of CTL epitopes. Since the proteasome is the major source of MHC class I-presented peptides, we analyzed the effect of proteasome inhibitors on the expression of surface HLA class I and the generation of EBV-derived CTL epitopes presented by the HLA-A2 and HLA-A11 alleles. Treatment with covalent and reversible inhibitors of the proteasome partially reduced the total and allele-specific expression of surface HLA class I in

EBV-carrying LCLs. HLA-A2 expression was also decreased by treatment with leupeptin and bestatin, while HLA-A11 expression was affected by treatment with phenanthroline. Despite their general inhibitory effect on HLA class I expression, all proteasome inhibitors tested enhanced the presentation of 2 subdominant HLA-A2 epitopes from EBV LMP1 and LMP2, while the presentation of the immunodominant HLA-A11-restricted epitope from EBNA4 was inhibited by MG132 and lactacystin and increased by ZL(3)VS. Treatment with ZL(3)VS restored the presentation of endogenously expressed EBNA4 in 1 HLA-A11-positive BL cell line. These findings suggest that specific inhibitors of the proteasome may be used to increase the antigenicity of **virus**-infected and malignant cells that are per se inefficient at generating particular CTL target epitopes.

CT Check Tags: Human; Support, Non-U.S. Gov't
***Antigen Presentation: DE, drug effects**
Cell Death: DE, drug effects
Cysteine Endopeptidases: ME, metabolism
Dose-Response Relationship, Drug
*Epitopes, T-Lymphocyte: IM, immunology
Epitopes, T-Lymphocyte: ME, metabolism
HLA-A Antigens: IM, immunology
HLA-A2 Antigen: IM, immunology
*Herpesvirus 4, Human: IM, immunology
*Multienzyme Complexes: AI, antagonists & inhibitors
Multienzyme Complexes: ME, metabolism
*Neoplasms: IM, immunology
*Neoplasms: VI, virology
***Protease Inhibitors: PD, pharmacology**
*T-Lymphocytes, Cytotoxic: IM, immunology
Time Factors
Tumor Cells, Cultured

CN 0 (Epitopes, T-Lymphocyte); 0 (HLA-A Antigens); 0 (HLA-A11); 0 (HLA-A2 Antigen); 0 (Multienzyme Complexes); 0 (**Protease Inhibitors**); EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.25.1 (proteasome endopeptidase complex)

L21 ANSWER 3 OF 12 MEDLINE on STN
AN 2000261640 MEDLINE
DN PubMed ID: 10799863
TI Sequential cleavage by metallopeptidases and proteasomes is involved in processing HIV-1 ENV epitope for endogenous MHC class I **antigen presentation.**
AU Lopez D; Gil-Torregrosa B C; Bergmann C; Del Val M
CS Centro Nacional de Biologia Fundamental, Instituto de Salud Carlos III, Madrid, Spain.
NC AI33314 (NIAID)
SO Journal of immunology (Baltimore, Md. : 1950), (2000 May 15) 164 (10) 5070-7.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; AIDS
EM 200006
ED Entered STN: 20000616
Last Updated on STN: 20030110
Entered Medline: 20000607
AB Antigenic peptides derived from viral proteins by multiple proteolytic cleavages are bound by MHC class I molecules and recognized by CTL. Processing predominantly takes place in the cytosol of infected cells by the action of proteasomes. To identify other proteases involved in the endogenous generation of viral epitopes, specifically those derived from

proteins routed to the secretory pathway, we investigated presentation of the HIV-1 ENV 10-mer epitope 318RGPGRAFVTI327 (p18) to specific CTL in the presence of diverse **protease inhibitors**. Both metalloproteinase and proteasome inhibitors decreased CTL recognition of the p18 epitope expressed from either native gp160 or from a chimera based on the hepatitis B **virus** secretory core protein as carrier protein. Processing of this epitope from both native ENV and the hepatitis B **virus** secretory core chimeric protein appeared to proceed by a TAP-dependent pathway that involved sequential cleavage by proteasomes and metallo-endopeptidases; however, other protease activities could replace the function of the lactacystin-sensitive proteasomes. By contrast, in a second TAP-independent pathway we detected no contribution of metallopeptidases for processing the ENV epitope from the chimeric protein. These results show that, in the classical TAP-dependent MHC class I pathway, endogenous Ag processing of viral proteins to yield the p18 10-mer epitope requires metallo-endopeptidases in addition to proteasomes.

CT Check Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
ATP-Binding Cassette Transporters: PH, physiology
Acetylcysteine: AA, analogs & derivatives
Acetylcysteine: PD, pharmacology
Animals
***Antigen Presentation**
Antigen Presentation: DE, drug effects
Cell Line, Transformed
Chimeric Proteins: IM, immunology
Chimeric Proteins: ME, metabolism
*Cysteine Endopeptidases: ME, metabolism
Cysteine Proteinase Inhibitors: PD, pharmacology
*Epitopes, T-Lymphocyte: ME, metabolism
*HIV Envelope Protein gp160: ME, metabolism
HIV-1: DE, drug effects
HIV-1: EN, enzymology
*HIV-1: IM, immunology
Hepatitis B e Antigens: GE, genetics
Hepatitis B e Antigens: ME, metabolism
*Histocompatibility Antigens Class I: ME, metabolism
Hydrolysis: DE, drug effects
Leupeptins: PD, pharmacology
*Metalloendopeptidases: ME, metabolism
Metalloendopeptidases: PH, physiology
Mice
Mice, Inbred BALB C
*Multienzyme Complexes: ME, metabolism
Pepstatins: PD, pharmacology
Peptide Fragments: AI, antagonists & inhibitors
Peptide Fragments: IM, immunology
Peptide Fragments: ME, metabolism
Protein Processing, Post-Translational: DE, drug effects
*Protein Processing, Post-Translational: IM, immunology
Signal Transduction: GE, genetics
Signal Transduction: IM, immunology
T-Lymphocytes, Cytotoxic: IM, immunology
T-Lymphocytes, Cytotoxic: ME, metabolism

RN 11076-29-2 (Streptomyces pepsin inhibitor); 133343-34-7 (lactacystin); 24365-47-7 (leupeptin); 39324-30-6 (pepstatin); 616-91-1 (Acetylcysteine)

CN 0 (ATP-Binding Cassette Transporters); 0 (Chimeric Proteins); 0 (Cysteine Proteinase Inhibitors); 0 (Epitopes, T-Lymphocyte); 0 (HIV Envelope Protein gp160); 0 (Hepatitis B e Antigens); 0 (Histocompatibility Antigens Class I); 0 (Leupeptins); 0 (Multienzyme Complexes); 0 (Pepstatins); 0 (Peptide Fragments); 0 (TAP1 protein, human); EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.24 (Metalloendopeptidases); EC 3.4.25.1

(proteasome endopeptidase complex)

L21 ANSWER 4 OF 12 MEDLINE on STN
AN 1999129194 MEDLINE
DN PubMed ID: 9930333
TI The proteasome system: a neglected tool for improvement of novel therapeutic strategies?.
AU Kloetzel P M
SO Gene therapy, (1998 Oct) 5 (10) 1297-8.
Journal code: 9421525. ISSN: 0969-7128.
CY ENGLAND: United Kingdom
DT Editorial
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990225
CT Check Tags: Human
Antigen Presentation
Epitopes
Genetic Engineering
Histocompatibility Antigens Class I: IM, immunology
*Neoplasms: DT, drug therapy
*Organelles: EN, enzymology
Peptide Hydrolases: IM, immunology
*Peptide Hydrolases: PH, physiology
***Protease Inhibitors: TU, therapeutic use**
Proteins: TU, therapeutic use
Vaccines, DNA
***Virus Diseases: DT, drug therapy**
CN 0 (Epitopes); 0 (Histocompatibility Antigens Class I); 0 (PSME1 protein, human); 0 (**Protease Inhibitors**); 0 (Proteins); 0 (Vaccines, DNA); EC 3.4 (Peptide Hydrolases)

L21 ANSWER 5 OF 12 MEDLINE on STN
AN 1999007277 MEDLINE
DN PubMed ID: 9789051
TI An inhibitor of HIV-1 protease modulates proteasome activity, **antigen presentation**, and T cell responses.
AU Andre P; Groettrup M; Klenerman P; de Giuli R; Booth B L Jr; Cerundolo V; Bonneville M; Jotereau F; Zinkernagel R M; Lotteau V
CS Institut Nationale de la Sante et de la Recherche Medicale U98X, Ecole Normale Superieure, 46 rue d'Italie, 69364 Lyon Cedex 07, France.
SO Proceedings of the National Academy of Sciences of the United States of America, (1998 Oct 27) 95 (22) 13120-4.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
EM 199811
ED Entered STN: 19990106
Last Updated on STN: 20000303
Entered Medline: 19981124
AB Inhibitors of the protease of HIV-1 have been used successfully for the treatment of HIV-1-infected patients and AIDS disease. We tested whether these protease inhibitory drugs exerted effects in addition to their antiviral activity. Here, we show in mice infected with lymphocytic choriomeningitis **virus** and treated with the HIV-1 protease inhibitor ritonavir a marked inhibition of antiviral cytotoxic T lymphocyte (CTL) activity and impaired major histocompatibility complex

class I-restricted epitope presentation in the absence of direct effects on lymphocytic choriomeningitis **virus** replication. A potential molecular target was found: ritonavir selectively inhibited the chymotrypsin-like activity of the 20S proteasome. In view of the possible role of T cell-mediated immunopathology in AIDS pathogenesis, the two mechanisms of action (i.e., reduction of HIV replication and impairment of CTL responses) may complement each other beneficially. Thus, the surprising ability of ritonavir to block the presentation of antigen to CTLs may possibly contribute to therapy of HIV infections but potentially also to the therapy of virally induced immunopathology, autoimmune diseases, and transplantation reactions.

CT Check Tags: Human; Support, Non-U.S. Gov't
Animals
*Cysteine Endopeptidases: ME, metabolism
Genes, MHC Class I: DE, drug effects
***HIV Protease Inhibitors: PD, pharmacology**
HIV Protease Inhibitors: TU, therapeutic use
HIV-1: EN, enzymology
Histocompatibility Antigens Class I: BI, biosynthesis
Immunity, Cellular
*Lymphocytic Choriomeningitis: DT, drug therapy
*Lymphocytic Choriomeningitis: IM, immunology
Lymphocytic choriomeningitis virus: IM, immunology
Mice
Mice, Inbred BALB C
Mice, Inbred C57BL
*Multienzyme Complexes: ME, metabolism
*Ritonavir: PD, pharmacology
Ritonavir: TU, therapeutic use
T-Lymphocytes: DE, drug effects
*T-Lymphocytes: IM, immunology
T-Lymphocytes, Cytotoxic: DE, drug effects
*T-Lymphocytes, Cytotoxic: IM, immunology

CN 0 (HIV **Protease Inhibitors**); 0 (Histocompatibility Antigens Class I); 0 (Multienzyme Complexes); 0 (Ritonavir); EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.25.1 (proteasome endopeptidase complex)

L21 ANSWER 6 OF 12 MEDLINE on STN
AN 1998209740 MEDLINE
DN PubMed ID: 9550370
TI Selective involvement of proteasomes and cysteine proteases in MHC class I **antigen presentation**.
AU Lopez D; Del Val M
CS Centro Nacional de Biologia Fundamental, Instituto de Salud Carlos III, Madrid, Spain.
SO Journal of immunology (Baltimore, Md. : 1950), (1997 Dec 15) 159 (12) 5769-72.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199804
ED Entered STN: 19980430
Last Updated on STN: 20000303
Entered Medline: 19980423

AB CTL recognize peptides derived from protein Ags bound to MHC-class I molecules. Proteasomes probably participate in the generation of these peptide epitopes. We investigated the role of proteasomes in the presentation of endogenously synthesized short viral proteins. To this end, we employed proteasome and cysteine **protease inhibitors** and two closely related recombinant vaccinia viruses

that code for 17- and 19-amino acid-long products encompassing murine CMV 9pp89 epitope. Presentation of both minigene products required processing to shorter peptides and was independent of ubiquitination. Proteasomes were necessary for processing the 17-mer product, and cysteine proteases were not required. In contrast, the 19-mer product could be processed in parallel either by proteasomes or by cysteine proteases independently. These results highlight the diversity of alternative processing pathways even for short peptidic Ags, provide evidence for the involvement of cysteine proteases in MHC class I presentation, and show that cleavage by cysteine proteases is governed by sequences flanking the epitope.

CT Check Tags: Support, Non-U.S. Gov't
Acetylcysteine: AA, analogs & derivatives
Acetylcysteine: PD, pharmacology
Amino Acid Sequence
Animals
***Antigen Presentation**
Antigen Presentation: DE, drug effects
Antigen Presentation: GE, genetics
Cell Line
*Cysteine Endopeptidases: IM, immunology
Cysteine Proteinase Inhibitors: PD, pharmacology
Cytomegalovirus: GE, genetics
Hepatitis B e Antigens: GE, genetics
*Histocompatibility Antigens Class I: ME, metabolism
Immediate-Early Proteins: GE, genetics
Immunodominant Epitopes: GE, genetics
Mice
Mice, Inbred BALB C
Molecular Sequence Data
*Multienzyme Complexes: IM, immunology
Mutagenesis, Insertional
T-Lymphocytes, Cytotoxic: EN, enzymology
T-Lymphocytes, Cytotoxic: IM, immunology
Vaccinia virus: GE, genetics
Vaccinia virus: IM, immunology

RN 133343-34-7 (lactacystin); 616-91-1 (Acetylcysteine)

CN 0 (Cysteine Proteinase Inhibitors); 0 (Hepatitis B e Antigens); 0 (Histocompatibility Antigens Class I); 0 (Immediate-Early Proteins); 0 (Immunodominant Epitopes); 0 (Multienzyme Complexes); 0 (cytomegalovirus immediate early phosphoprotein pp89); EC 3.4.22 (Cysteine Endopeptidases); EC 3.4.25.1 (proteasome endopeptidase complex)

L21 ANSWER 7 OF 12 MEDLINE on STN

AN 97461594 MEDLINE

DN PubMed ID: 9314557

TI Two novel routes of transporter associated with antigen processing (TAP)-independent major histocompatibility complex class I antigen processing.

AU Snyder H L; Bacik I; Bennink J R; Kearns G; Behrens T W; Bachi T; Orlowski M; Yewdell J W

CS Laboratory of Viral Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892-0440, USA.

SO Journal of experimental medicine, (1997 Oct 6) 186 (7) 1087-98.
Journal code: 2985109R. ISSN: 0022-1007.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; AIDS

EM 199711

ED Entered STN: 19971224
Last Updated on STN: 20000303
Entered Medline: 19971113

AB Jaw1 is an endoplasmic reticulum (ER) resident protein representative of a class of proteins post translationally inserted into membranes via a type II membrane anchor (cytosolic NH2 domain, lumenal COOH domain) in a translocon-independent manner. We found that Jaw1 can efficiently deliver a COOH-terminal antigenic peptide to class I molecules in transporter associated with antigen processing (TAP)-deficient cells or cells in which TAP is inactivated by the ICP47 protein. Peptide delivery mediated by Jaw1 to class I molecules was equal or better than that mediated by the adenovirus E3/19K glycoprotein signal sequence, and was sufficient to enable cytofluorographic detection of newly recruited thermostabile class I molecules at the surface of TAP-deficient cells. Deletion of the transmembrane region retargeted Jaw1 from the ER to the cytosol, and severely, although incompletely, abrogated its TAP-independent peptide carrier activity. Use of different **protease inhibitors** revealed the involvement of a nonproteasomal protease in the TAP-independent activity of cytosolic Jaw1. These findings demonstrate two novel TAP-independent routes of antigen processing; one based on highly efficient peptide liberation from the COOH terminus of membrane proteins in the ER, the other on delivery of a cytosolic protein to the ER by an unknown route.

CT Check Tags: Human
***Antigen Presentation: IM, immunology**
Blotting, Western
CD8-Positive T-Lymphocytes: IM, immunology
*Carrier Proteins: ME, metabolism
Cell Line
Chimeric Proteins
Cytosol: ME, metabolism
Endopeptidases: ME, metabolism
Endoplasmic Reticulum: EN, enzymology
Gene Expression Regulation
Hela Cells
*Histocompatibility Antigens Class I: IM, immunology
Membrane Proteins: GE, genetics
Membrane Proteins: IM, immunology
*Membrane Proteins: ME, metabolism
Microscopy, Immunoelectron
Peptides: ME, metabolism
Protease Inhibitors: PD, pharmacology
Transformation, Genetic
Vaccinia virus: GE, genetics
Viral Proteins: GE, genetics
Viral Proteins: ME, metabolism

CN 0 (Carrier Proteins); 0 (Chimeric Proteins); 0 (Histocompatibility Antigens Class I); 0 (JAW1 gene product); 0 (Membrane Proteins); 0 (Peptides); 0 (**Protease Inhibitors**); 0 (Viral Proteins); EC 3.4.- (Endopeptidases)

L21 ANSWER 8 OF 12 MEDLINE on STN
AN 97303796 MEDLINE
DN PubMed ID: 9160098
TI MHC class I presentation of live and heat-inactivated Sendai **virus** antigen in T2Kb cells depends on an intracellular compartment with endosomal characteristics.
AU Liu T; Zhou X; Abdel-Motal U M; Ljunggren H G; Jondal M
CS Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, Sweden.
SO Scandinavian journal of immunology, (1997 May) 45 (5) 527-33.
Journal code: 0323767. ISSN: 0300-9475.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199706
ED Entered STN: 19970620
Last Updated on STN: 19970620
Entered Medline: 19970612
AB T2Kb cells, which do not express TAP1/2 peptide transporters or the low molecular weight protein 2/7 (LMP2/7) proteasomal subunits, can still process and present both live and heat-inactivated Sendai **virus** (SV). As this operation may also reflect the existence of an alternative processing pathway in normal antigen-presenting cells (APC), the authors have characterized it using intracellular inhibitors and anti-Kb monoclonal antibodies (MoAbs). From the results with lipophilic amines (ammonium chloride, methylamine and chloroquine), cytoskeletal inhibitors (cytochalasin B and vinblastine), and an endoprotease inhibitor (phenylmethylsulfonyl fluoride, PMSF), the authors conclude that the processing of SV antigen in T2Kb cells has endosomal characteristics depending on cellular activities such as uptake, vesicular transport and intracellular-vesicular proteolysis. In addition, internalized 'empty' Kb molecules derived from the T2Kb cell surface appeared to be involved in the presentation of SV antigen, as demonstrated by a protocol using a combination of the Golgi inhibitor brefeldin A(BFA) and anti-Kb antibodies. The results thus indicate that T2Kb cells process SV antigen in an endosomal-like compartment which contain recycling 'empty' Kb molecules.
CT Check Tags: Female; Support, Non-U.S. Gov't
Amines: PD, pharmacology
Animals
Antibodies, Monoclonal
***Antigen Presentation**
Antigen Presentation: DE, drug effects
*Antigens, Viral
Cell Compartmentation
Cell Line
Cytochalasin B: PD, pharmacology
Cytoskeleton: DE, drug effects
Endosomes: DE, drug effects
*Endosomes: IM, immunology
*H-2 Antigens: ME, metabolism
Heat
Mice
Mice, Inbred C57BL
Phenylmethylsulfonyl Fluoride: PD, pharmacology
Protease Inhibitors: PD, pharmacology
*Respirovirus: IM, immunology
Vinblastine: PD, pharmacology
RN 14930-96-2 (Cytochalasin B); 329-98-6 (Phenylmethylsulfonyl Fluoride); 865-21-4 (Vinblastine)
CN 0 (Amines); 0 (Antibodies, Monoclonal); 0 (Antigens, Viral); 0 (H-2 Antigens); 0 (H-2Kb); 0 (**Protease Inhibitors**)

L21 ANSWER 9 OF 12 MEDLINE on STN
AN 96005067 MEDLINE
DN PubMed ID: 7561783
TI Vaccinia **virus** serpins B13R and B22R do not inhibit **antigen presentation** to class I-restricted cytotoxic T lymphocytes.
AU Blake N W; Kettle S; Law K M; Gould K; Bastin J; Townsend A R; Smith G L
CS Sir William Dunn School of Pathology, University of Oxford, UK.
SO Journal of general virology, (1995 Sep) 76 (Pt 9) 2393-8.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals
EM 199511
ED Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951113
AB Vaccinia **virus** (VV) inhibits the presentation of certain epitopes from influenza **virus** nucleoprotein (NP), haemagglutinin (HA) and non-structural 1 (NS1) proteins to CD8+ cytotoxic T lymphocytes (CTL) by an unknown mechanism. We have investigated whether VV genes B13R and B22R, which encode proteins with amino acid similarity to serine **protease inhibitors** (serpins), are involved in this process. Recombinant VVs were constructed which express influenza **virus** proteins HA, NP or NS1 and which lack serpin gene B13R or both B13R and B22R. The lysis of cells infected with these viruses by influenza **virus**-specific CD8+ CTL was compared to the lysis of cells infected with viruses expressing both the influenza proteins and the serpin genes. Cytotoxicity assays showed that deletion of the VV serpin genes B13R and B22R and other genes between B13R and B24R did not increase the level of lysis, indicating that these genes are not involved in inhibition of **antigen presentation** of the epitopes tested.
CT Check Tags: Support, Non-U.S. Gov't
Animals
***Antigen Presentation**
Cell Line
Hemagglutinin Glycoproteins, Influenza Virus
Hemagglutinins, Viral: GE, genetics
Hemagglutinins, Viral: IM, immunology
Histocompatibility Antigens Class I
Influenza A virus: GE, genetics
Influenza A virus: IM, immunology
Mice
Nucleoproteins: GE, genetics
Nucleoproteins: IM, immunology
Recombinant Fusion Proteins: ME, metabolism
Serpins: GE, genetics
*Serpins: IM, immunology
*T-Lymphocytes, Cytotoxic: IM, immunology
Vaccinia virus: GE, genetics
***Vaccinia virus: IM, immunology**
Viral Core Proteins: GE, genetics
Viral Core Proteins: IM, immunology
Viral Nonstructural Proteins: GE, genetics
Viral Nonstructural Proteins: IM, immunology
Viral Proteins: GE, genetics
*Viral Proteins: IM, immunology
CN 0 (Hemagglutinin Glycoproteins, Influenza **Virus**); 0 (Hemagglutinins, Viral); 0 (Histocompatibility Antigens Class I); 0 (INS1 protein, influenza **virus**); 0 (Nucleoproteins); 0 (Recombinant Fusion Proteins); 0 (Serpins); 0 (Viral Core Proteins); 0 (Viral Nonstructural Proteins); 0 (Viral Proteins); 0 (influenza A **virus** nucleoprotein)

L21 ANSWER 10 OF 12 MEDLINE on STN
AN 95197153 MEDLINE
DN PubMed ID: 7890301
TI Modulation of antigen processing and presentation by covalently linked complement C3b fragment.
AU Jacquier-Sarlin M R; Gabert F M; Villiers M B; Colomb M G
CS Unite INSERM 238, Centre d'Etudes Nucleaires de Grenoble, France.
SO Immunology, (1995 Jan) 84 (1) 164-70.

Journal code: 0374672. ISSN: 0019-2805.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199504
ED Entered STN: 19950427
Last Updated on STN: 19970203
Entered Medline: 19950418
AB Ligands such as complement fragments (C3, C4), IgG or alpha 2-macroglobulin, which bind antigen (Ag) before their uptake by antigen-presenting cells (APC), are likely to modulate the different steps of Ag processing and presentation. These ligands contribute to internalization and endosomal targeting of Ag; they also influence its processing and, consequently, the binding of resulting peptides to major histocompatibility complex (MHC) class II molecules before presentation to T cells. Complement protein C3 contains, like other members of the alpha 2-macroglobulin family, an intrachain thiolester bond. Conformational alteration or limited proteolysis of C3 into C3b leads to breaking of the thiolester with transient capacity of the revealed carbonyl group to esterify hydroxyl groups of Ag. Ester-linked complexes including tetanus toxin (TT) and C3b were prepared to analyse the influence of bound C3b on TT processing and presentation by APC. Covalent binding of C3b to TT resulted in increased and prolonged stimulation of specific T-cell proliferation. This effect was observed with non-specific B cells, as well as with a TT-specific B-cell clone, as APC. On the other hand, SDS-PAGE analysis of proteolysates of TT or C3b-TT, obtained with endosome/lysosome-enriched subcellular fractions prepared from human Epstein-Barr **virus** (EBV)-transformed B cells, indicated a delay of TT proteolysis when TT was associated to C3b. Treatment of APC with **protease inhibitors**, before and during exposure of the cells to Ag, resulted in differences in the inhibition of TT and C3b-TT proteolysis. Using purified cathepsins B and D, we demonstrated that covalent binding of C3b to TT totally abolished TT proteolysis by cathepsin D, while proteolysis by cathepsin B was preserved. This finding and the absence of cathepsin B in endosomes may explain a delay in TT processing when it is associated to C3b. Confirming these data, presentation by formaldehyde-fixed cells of C3b-TT proteolysates showed higher stimulation of specific T-cell clones than formaldehyde-fixed TT proteolysates.
CT Check Tags: Human
***Antigen Presentation: IM, immunology**
*Antigen-Presenting Cells: IM, immunology
*Antigenic Modulation: IM, immunology
B-Lymphocytes: IM, immunology
Cathepsin B: ME, metabolism
Cathepsin D: ME, metabolism
Cell Line
*Complement 3b: ME, metabolism
Electrophoresis, Polyacrylamide Gel
Lymphocyte Activation
Protein Binding
T-Lymphocytes: CY, cytology
T-Lymphocytes: IM, immunology
*Tetanus Toxin: ME, metabolism
Time Factors
RN 80295-43-8 (Complement 3b)
CN 0 (Tetanus Toxin); EC 3.4.22.1 (Cathepsin B); EC 3.4.23.5 (Cathepsin D)

L21 ANSWER 11 OF 12 MEDLINE on STN
AN 93203607 MEDLINE
DN PubMed ID: 7681081

TI Comparison of **antigen presentation** of influenza A nucleoprotein expressed in attenuated AroA- Salmonella typhimurium with that of live **virus**.

AU Brett S J; Rhodes J; Liew F Y; Tite J P
CS Department of Cell Biology, Wellcome Research Laboratories, Beckenham, Kent, UK.

SO Journal of immunology (Baltimore, Md. : 1950), (1993 Apr 1) 150 (7) 2869-84.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199304
ED Entered STN: 19930507
Last Updated on STN: 19990129
Entered Medline: 19930420

AB Rationally attenuated strains of Salmonella expressing foreign proteins represent a potentially important vaccine delivery system. The characteristics of Ag presentation of influenza nucleoprotein expressed in an AroA- strain of Salmonella typhimurium (SL3262-pNP-2) have therefore been compared with those of soluble purified nucleoprotein (NP) and infectious influenza **virus**. This represents three distinct modes of internalization of the same protein into APC. Human monocytes and the monocytic leukemia cell line THP-1 infected with SL3261-pNP-2 were found to present several different epitopes from NP to human CD4+ class II-restricted T lymphocytes. Ag presentation to these T cell clones was enhanced by pretreatment of THP-1 cells with IFN-gamma but not TNF-alpha. Bacterial phagocytosis and Ag presentation of NP were increased after opsonization of Salmonella with immune serum. Macrophages infected with SL3261-pNP-2 were unable to present NP to class I-restricted T cells. In contrast, cells infected with live influenza **virus**, although recognized by NP-specific class I-restricted CTL, were inefficiently recognized by NP-specific class II-restricted T cells. Ag presentation to CD4+ T cell clones by monocytes of SL3261-pNP-2, purified recombinant NP, and live influenza **virus**, but not the synthetic peptide 206-229, was inhibited by chloroquine and the **protease inhibitors** pepstatin A and leupeptin, suggesting that the major route of processing in each case was via the exogenous pathway. T cell recognition of NP via all of these Ag delivery systems was also abrogated by cycloheximide and brefeldin A treatment, indicating a requirement for recently synthesized MHC class II molecules in presentation of whole NP after processing but not for the corresponding synthetic peptide.

CT Check Tags: Comparative Study; Female; Human
*Alkyl and Aryl Transferases
Animals
*Antigen-Presenting Cells: IM, immunology
Antigens, Differentiation, T-Lymphocyte: IM, immunology
*Bacterial Proteins: IM, immunology
Brefeldin A
Cycloheximide: PD, pharmacology
Cyclopentanes: PD, pharmacology
Epitopes: IM, immunology
Genetic Vectors
Histocompatibility Antigens Class I: IM, immunology
***Influenza A virus: IM, immunology**
Influenza A virus: PY, pathogenicity
Kinetics
Macrophages: IM, immunology
Mice
Mice, Inbred BALB C
Monocytes: IM, immunology

Monocytes: MI, microbiology
*Nucleoproteins: IM, immunology
Phagocytosis
Protease Inhibitors: PD, pharmacology
Salmonella typhimurium: GE, genetics
*Salmonella typhimurium: IM, immunology
Salmonella typhimurium: PY, pathogenicity
Tumor Cells, Cultured
*Viral Core Proteins: IM, immunology
Virulence

RN 20350-15-6 (Brefeldin A); 66-81-9 (Cycloheximide)

CN 0 (Antigens, Differentiation, T-Lymphocyte); 0 (Bacterial Proteins); 0 (Cyclopentanes); 0 (Epitopes); 0 (Genetic Vectors); 0 (Histocompatibility Antigens Class I); 0 (Nucleoproteins); 0 (**Protease Inhibitors**); 0 (Viral Core Proteins); 0 (influenza A **virus** nucleoprotein); EC 2.5 (Alkyl and Aryl Transferases); EC 2.5.1.19 (3-phosphoshikimate 1-carboxyvinyltransferase)

L21 ANSWER 12 OF 12 MEDLINE on STN

AN 91363237 MEDLINE

DN PubMed ID: 1888663

TI Inhibition of the presentation of dengue **virus** antigen by macrophages to B cells by serine-**protease inhibitors**.

AU Rizvi N; Chaturvedi U C; Mathur A

CS Postgraduate Department of Microbiology, K.G. Medical College, Lucknow, India.

SO International journal of experimental pathology, (1991 Feb) 72 (1) 23-9. Journal code: 9014042. ISSN: 0959-9673.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199110

ED Entered STN: 19911103
Last Updated on STN: 20000303
Entered Medline: 19911011

AB It has been shown that macrophages (M phi) process dengue type 2 **virus** (DV) antigen and present it to B cells leading to their clonal expansion as shown by DV-specific IgM antibody plaque-forming cell (PFC) count in spleen. The present study was undertaken to find out the nature of enzymes responsible for the processing of DV antigen in M phi. DV-pulsed M phi were treated with seven different **protease inhibitors** and then assayed for **antigen presentation** to B cells. It was observed that maximum inhibition occurred by treatment of M phi with PMSF, a serine-protease inhibitor. The effect of PMSF was dose dependent and was abolished by using predigested antigen. PMSF inhibited presentation of DV and sheep RBC antigens but had no effect on presentation of bovine serum albumin which does not require processing. The results thus identify the serine group of proteases as the main enzymes involved in processing the DV antigen in M phi.

CT Animals
Antigen-Presenting Cells: EN, enzymology
*Antigens, Viral: IM, immunology
B-Lymphocytes: IM, immunology
***Dengue Virus: IM, immunology**
*Macrophages: EN, enzymology
Macrophages: IM, immunology

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d his

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L1             0 S IND7312
L2           110 S CMV PP65
L3          3417 S ANTI APOPTOTIC
L4             0 S L2 AND L3

     FILE 'CA' ENTERED AT 14:56:32 ON 28 JUN 2004
L5             0 S L3 AND L2

     FILE 'BIOSIS' ENTERED AT 14:56:56 ON 28 JUN 2004
L6             1 S L3 AND L2

     FILE 'CAPLUS' ENTERED AT 14:57:45 ON 28 JUN 2004
L7             0 S L3 AND L2

     FILE 'SCISEARCH' ENTERED AT 14:57:58 ON 28 JUN 2004
L8             2 S L3 AND L2

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     BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
     CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DISSABS,
     DDFB, DDFU, DGENE, DRUGB, DRUGMONOG2, ...' ENTERED AT 14:59:15 ON 28 JUN
     2004
               SEA L3 AND L2
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              1    FILE BIOSIS
              2    FILE SCISEARCH
              1    FILE USPATFULL
L9             QUE L3 AND L2
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     FILE 'USPATFULL' ENTERED AT 15:00:12 ON 28 JUN 2004
L10            1 S L3 AND L2

     FILE 'MEDLINE' ENTERED AT 15:00:48 ON 28 JUN 2004
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First Hit

Generate Collection | Print

L7: Entry 1 of 4 File: DWPI Sep 19, 2002

DERWENT-ACC-NO: 2002-740762
DERWENT-WEEK: 200433

TITLE: Use of anti-apoptotic reagent for preservation of antigen presentation on a virally infected mammalian cell

INVENTOR: AJA, T; CHING, B W ; GLADSTONE, P L

PRIORITY-DATA: 2001US-272750P (March 2, 2001), 2002US-0087607 (March 1, 2002)

Search Selected | Search ALL | Clear

PATENT-FAMILY:

	PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
☐	AU 2002245649 A1	September 19, 2002		000	C07K000/00
☐	WO 200270544 A2	September 12, 2002	E	148	C07K000/00
☐	US 20030039661 A1	February 27, 2003		000	A61K039/12

INT-CL (IPC): A61 K 39/12; A61 K 39/21; A61 K 39/29; C07 K 0/00

ABSTRACTED-PUB-NO: WO 200270544A
BASIC-ABSTRACT:

NOVELTY - Preservation of antigen presentation on a virally infected mammalian cell involves contacting a population of partly virally infected mammalian cells with an anti-apoptotic reagent.

ACTIVITY - None given.

MECHANISM OF ACTION - Apoptosis including interleukin-1 beta -converting enzyme (ICE)/CED-3 inhibitor.

USE - For preserving antigen presentation on virally infected (particularly herpes, HIV, cytomegalovirus and hepatitis) mammalian cell (preferably peripheral blood leukocytes, neutrophils and granulocytes) (claimed).

ADVANTAGE - The apoptotic inhibitors preserve antigen positivity for a longer time (preferably 72 hours) and therefore increase sample stability so that more robust CMV antigenemia assay can be carried out in centralized laboratories. The method allows longer period of time between collection and processing of samples.

ABSTRACTED-PUB-NO: WO 200270544A
EQUIVALENT-ABSTRACTS:

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 10 of 21 returned.

☐ 1. Document ID: US 6723563 B2

L4: Entry 1 of 21 File: USPT Apr 20, 2004

US-PAT-NO: 6723563
DOCUMENT-IDENTIFIER: US 6723563 B2

TITLE: Hematology reference control

DATE-ISSUED: April 20, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ryan; Wayne L.	Omaha	NE		

US-CL-CURRENT: 436/10; 422/73, 435/2, 436/17, 436/174, 436/63, 436/8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 2. Document ID: US 6562621 B1

L4: Entry 2 of 21 File: USPT May 13, 2003

US-PAT-NO: 6562621
DOCUMENT-IDENTIFIER: US 6562621 B1

TITLE: Method of using fish ovarian fluid for culture and preservation of mammalian cells

DATE-ISSUED: May 13, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Sawyer; Evelyn S.	Arundel	ME		
Sawyer; Philip J.	Arundel	ME		
Janmey; Paul A.	Arundel	ME		

US-CL-CURRENT: 435/408; 424/537, 424/559, 435/1.1, 435/2, 435/374, 435/391

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 5 of 5 returned.

☐ 1. Document ID: US 6693096 B2

L3: Entry 1 of 5 File: USPT Feb 17, 2004

US-PAT-NO: 6693096
DOCUMENT-IDENTIFIER: US 6693096 B2

TITLE: Treatment of inflammation-associated disorders using interleukin-1.beta.-converting enzyme (ICE)/CED-3 family inhibitors

DATE-ISSUED: February 17, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fritz; Lawrence C.	Rancho Santa Fe	CA		
Tomaselli; Kevin J.	San Diego	CA		
Karanewsky; Donald S.	Escondido	CA		
Linton; Steven D.	San Diego	CA		
Bai; Xu	Carlsbad	CA		

US-CL-CURRENT: 514/212.05, 514/419

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

☐ 2. Document ID: US 6610683 B2

L3: Entry 2 of 5 File: USPT Aug 26, 2003

US-PAT-NO: 6610683
DOCUMENT-IDENTIFIER: US 6610683 B2

TITLE: Treatment of infectious disease using interleukin-1.beta.-converting enzyme (ICE)/CED-3 family inhibitors

DATE-ISSUED: August 26, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fritz; Lawrence C.	Rancho Santa Fe	CA		
Tomaselli; Kevin J.	San Diego	CA		
Karanewsky; Donald S.	Escondido	CA		

Linton; Steven D.	San Diego	CA
Bai; Xu	Carlsbad	CA

US-CL-CURRENT: 514/212.05; 514/415, 514/419

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | Claims | KWIC | Draw De

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☐ 3. Document ID: US 6531467 B2

L3: Entry 3 of 5 File: USPT Mar 11, 2003

US-PAT-NO: 6531467
DOCUMENT-IDENTIFIER: US 6531467 B2
**** See image for Certificate of Correction ****

TITLE: Inhibition of inflammation using interleukin-1.beta.-converting enzyme (ICE)/CED-3 family inhibitors

DATE-ISSUED: March 11, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fritz; Lawrence C.	Rancho Santa Fe	CA		
Tomaselli; Kevin J.	San Diego	CA		
Karanewsky; Donald S.	Escondido	CA		
Linton; Steven D.	San Diego	CA		
Bai; Xu	Carlsbad	CA		
Montisano; Dominic F.	San Diego	CA		
Higgins; David	San Diego	CA		

US-CL-CURRENT: 514/212.05; 514/419

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | Claims | KWIC | Draw De

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☐ 4. Document ID: US 6528506 B2

L3: Entry 4 of 5 File: USPT Mar 4, 2003

US-PAT-NO: 6528506
DOCUMENT-IDENTIFIER: US 6528506 B2
**** See image for Certificate of Correction ****

TITLE: Inhibition of apoptosis using interleukin-1.beta.-converting enzyme (ICE)/CED-3 family inhibitors

DATE-ISSUED: March 4, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fritz; Lawrence C.	Rancho Santa Fe	CA		

Tomaselli; Kevin J.	San Diego	CA
Karanewski; Donald S.	Escondido	CA
Linton; Steven D.	San Diego	CA
Bai; Xu	Carlsbad	CA

US-CL-CURRENT: 514/212.05; 514/419

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw D

☐ 5. Document ID: US 6200969 B1

L3: Entry 5 of 5 File: USPT Mar 13, 2001

US-PAT-NO: 6200969
DOCUMENT-IDENTIFIER: US 6200969 B1
**** See image for Certificate of Correction ****

TITLE: Inhibition of apoptosis using interleukin-1.beta.-converting enzyme (ICE)/CED-3 family inhibitors

DATE-ISSUED: March 13, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fritz; Lawrence C.	Rancho Santa Fe	CA		
Tomaselli; Kevin J.	San Diego	CA		
Karanewski; Donald S.	Escondido	CA		
Linton; Steven D.	San Diego	CA		
Bai; Xu	Carlsbad	CA		

US-CL-CURRENT: 514/212.05; 514/419

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw D

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
L1 and survival	5

Display Format: CIT Change Format

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☐ 3. Document ID: US 6512167 B1

L4: Entry 3 of 21 File: USPT Jan 28, 2003

US-PAT-NO: 6512167
DOCUMENT-IDENTIFIER: US 6512167 B1

TITLE: Hybrid maize seed and plant RPG824

DATE-ISSUED: January 28, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Carolo; Pierre	Vendome			FR

US-CL-CURRENT: 800/320.1; 435/412, 435/421, 435/424, 435/430, 435/430.1, 435/468, 800/265, 800/266, 800/268, 800/271, 800/275, 800/278

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 4. Document ID: US 6490588 B2

L4: Entry 4 of 21 File: USPT Dec 3, 2002

US-PAT-NO: 6490588
DOCUMENT-IDENTIFIER: US 6490588 B2

TITLE: Method of searching novel ligand compounds from three-dimensional structure database

DATE-ISSUED: December 3, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Itai; Akiko	Tokyo		113	JP
Mizutani; Miho	Tokyo		112	JP

US-CL-CURRENT: 707/10

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 5. Document ID: US 6389378 B2

L4: Entry 5 of 21 File: USPT May 14, 2002

US-PAT-NO: 6389378
DOCUMENT-IDENTIFIER: US 6389378 B2
** See image for Certificate of Correction **

TITLE: Method of searching novel ligand compounds from three-dimensional structure

database

DATE-ISSUED: May 14, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Itai; Akiko	Tokyo			JP
Mizutani; Miho	Tokyo			JP

US-CL-CURRENT: 703/11; 702/27, 707/104.1, 707/6

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

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☐ 6. Document ID: US 6280729 B1

L4: Entry 6 of 21 File: USPT Aug 28, 2001

US-PAT-NO: 6280729
DOCUMENT-IDENTIFIER: US 6280729 B1

TITLE: Preparation of factor IX

DATE-ISSUED: August 28, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Huang; Chin C.	Bourbonnais	IL		
Enkoji; Takashi	Park Forest	IL		
Ho; Laura	Bourbonnais	IL		
Kleszynski; Richard R.	St. Anne	IL		
Weeks; Richard L.	Kankakee	IL		
Feldman; Fred	Frankfort	IL		

US-CL-CURRENT: 424/94.64; 514/8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw De

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☐ 7. Document ID: US 6162242 A

L4: Entry 7 of 21 File: USPT Dec 19, 2000

US-PAT-NO: 6162242
DOCUMENT-IDENTIFIER: US 6162242 A

TITLE: Selective photodynamic treatment

DATE-ISSUED: December 19, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY

Peyman; Gholam A. New Orleans LA 70124

US-CL-CURRENT: 607/88; 128/898

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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☐ 8. Document ID: US 6114117 A

L4: Entry 8 of 21 File: USPT Sep 5, 2000

US-PAT-NO: 6114117
DOCUMENT-IDENTIFIER: US 6114117 A

TITLE: Homogeneous diagnostic assay method utilizing simultaneous target and signal amplification

DATE-ISSUED: September 5, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hepp; Jozsef	Camarillo	CA		
Lengyel; Zsolt	Camarillo	CA		
Pande; Rajiv	Ventura	CA		
Botyanszki; Janos	Camarillo	CA		
Sahin-Toth; Miklos	Camarillo	CA		

US-CL-CURRENT: 435/6; 536/23.1, 536/24.3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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☐ 9. Document ID: US 6063909 A

L4: Entry 9 of 21 File: USPT May 16, 2000

US-PAT-NO: 6063909
DOCUMENT-IDENTIFIER: US 6063909 A
**** See image for Certificate of Correction ****

TITLE: Preparation of factor IX

DATE-ISSUED: May 16, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Huang; Chin C.	Bourbonnais	IL		
Takashi; Enkoji	Park Forest	IL		
Ho; Laura	Bourbonnais	IL		
Kleszynski; Richard R.	St. Anne	IL		
Weeks; Richard L.	Kankakee	IL		

Feldman; Fred Frankfort IL

US-CL-CURRENT: 530/412; 530/381, 530/413

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 10. Document ID: US 6016712 A

L4: Entry 10 of 21 File: USPT Jan 25, 2000

US-PAT-NO: 6016712
DOCUMENT-IDENTIFIER: US 6016712 A

TITLE: Device for receiving and processing a sample

DATE-ISSUED: January 25, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Warden; Laurence	Poway	CA		
Kaplan; David E.	Carlsbad	CA		

US-CL-CURRENT: 73/864.21; 73/864.22

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
protease inhibitor and preserving.clm.	21

Display Format: CIT Change Format

Previous Page Next Page Go to Doc#

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Monday, June 28, 2004

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☐	L15	wo2002070544	0
		DB=DWPI; PLUR=YES; OP=ADJ	
☐	L14	wo2002070544	0
☐	L13	wo2002070544A2	0
☐	L12	wo2002070544A3	0
		DB=PGPB; PLUR=YES; OP=ADJ	
☐	L11	protease inhibitor and preserving.clm.	20
		DB=EPAB; PLUR=YES; OP=ADJ	
☐	L10	Aja .in.	2
☐	L9	WO-200270544-A2.did.	0
		DB=PGPB; PLUR=YES; OP=ADJ	
☐	L8	US-20030039661-A1.did.	1
		DB=DWPI; PLUR=YES; OP=ADJ	
☐	L7	Aja .in.	4
☐	L6	Aja T J.in.	0
		DB=USPT; PLUR=YES; OP=ADJ	
☐	L5	6207817.pn.	1
☐	L4	protease inhibitor and preserving.clm.	21
☐	L3	protease inhibitor and preserving	537
☐	L2	protease inhibitor	10239
☐	L1	protease	33269

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